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EGFR Signal Induced-Nuclear Translocation of Beta-catenin and PKM2 Promotes HCC Malignancy and Indicates Early Recurrence After Curative Resection

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Abstract : Early recurrence (ER) (< 1 year) after liver resection is one of the most important factors that impacts the prognosis of patients with hepatocellular carcinoma (HCC). However, the molecular mechanisms and predictive indexes of ER after curative resection remain largely unknown. The present study aimed to exploit the role of EGFR signaling in EMT and early recurrence of HCC after curative resection and elucidate the molecular mechanisms. Our results showed that nuclear beta-catenin / PKM2 was a independent predictor of early recurrence after curative resection in EGFR-overexpressed HCC. Mechanistic investigation indicated that nuclear accumulation of beta-catenin and PKM2 induced by EGFR signal promoted HCC cell invasion and proliferation, which were required for early recurrence of HCC. These effects were mediated by PI3K/AKT and ERK pathways rather than the canonical Wnt signaling. In conclusions, EGFR signal induced-nuclear translocation of beta-catenin and PKM2 promotes HCC malignancy and indicates early recurrence after curative resection.

Keywords : beta-catenin, early recurrence, hepatocellular carcinoma, malignancy, PKM2
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